LG-4878 REV. 9/89

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DUPONT

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Wilmington, Delaware 19898

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INIT 10-27-92
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October 15, 1992

Document Processing Center (TS-790)
Office of Pollution Prevention and Toxics
Environmental Protection Agency
401 M Street., S.W.
Washington, D.C. 20460
Attn: Section 8(e) Coordinator (CAP Agreement)

Dear Coordinator:

8ECAP-0025

On behalf of the Regulatee and pursuant to Unit II B.1.b. and Unit II C of the 6/28/91CAP Agreement, E.I. Du Pont de Nemours and Co. hereby submits (*in triplicate*) the attached studies. Submission of this information is voluntary and is occasioned by unilateral changes in EPA's standard as to what EPA now considers as reportable information. Regulatee's submission of information is made solely in response to the new EPA §8(e) reporting standards and is not an admission: (1) of TSCA violation or liability; (2) that Regulatee's activities with the study compounds reasonably support a conclusion of substantial health or environmental risk or (3) that the studies themselves reasonably support a conclusion of substantial health or environmental risk.

The "Reporting Guide" creates new TSCA 8(e) reporting criteria which were not previously announced by EPA in its 1978 Statement of Interpretation and Enforcement Policy, 43 Fed Reg 11110 (March 16, 1978). The "Reporting Guide states criteria which expands upon and conflicts with the 1978 Statement of Interpretation. Absent amendment of the Statement of Interpretation, the informal issuance of the "Reporting Guide" raises significant due processes issues and clouds the appropriate reporting standard by which regulated persons can assure TSCA Section 8(e) compliance.

For Regulatee,

Mark H. Christman
Counsel
Legal D-7158
1007 Market Street
Wilmington, DE 19898
(302) 774-6443

RECEIVED 8/05/95

ATTACHMENT 1

Submission of information is made under the 6/28/91 CAP Agreement, Unit II. This submission is made voluntarily and is occasioned by recent changes in EPA's TSCA §8(e) reporting standard; such changes made, for the first time in 1991 and 1992 without prior notice and in violation of Regulatee's constitutional due process rights. Regulatee's submission of information under this changed standard is not a waiver of its due process rights; an admission of TSCA violation or liability, or an admission that Regulatee's activities with the study compounds reasonably support a conclusion of substantial risk to health or to the environment. Regulatee has historically relied in good faith upon the 1978 Statement of Interpretation and Enforcement Policy criteria for determining whether study information is reportable under TSCA §8(e), 43 Fed Reg 11110 (March 16, 1978). EPA has not, to date, amended this Statement of Interpretation.

After CAP registration, EPA provided the Regulatee the June 1, 1991 "TSCA Section 8(e) Reporting Guide". This "Guide" has been further amended by EPA, EPA letter, April 10, 1992. EPA has not indicated that the "Reporting Guide" or the April 1992 amendment supersedes the 1978 Statement of Interpretation. The "Reporting Guide" and April 1992 amendment substantively lowers the Statement of Interpretation 's TSCA §8(e) reporting standard[2]. This is particularly troublesome as the "Reporting Guide" states criteria, applied retroactively, which expands upon and conflicts with the Statement of Interpretation.[3] Absent amendment of the Statement of Interpretation, the informal issuance of the "Reporting Guide" and the April 1992 amendment clouds the appropriate standard by which regulated persons must assess information for purposes of TSCA §8(e).

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[2]In sharp contrast to the Agency's 1977 and 1978 actions to soliciting public comment on the proposed and final §8(e) Policy, EPA has unilaterally pronounced §8(e) substantive reporting criteria in the 1991 Section 8(e) Guide without public notice and comment. See 42 Fed Reg 45362 (9/9/77), "Notification of Substantial Risk under Section 8(e): Proposed Guidance".

[3]A comparison of the 1978 Statement of Interpretation and the 1992 "Reporting Guide" is a appended.

Throughout the CAP, EPA has mischaracterized the 1991 guidance as reflecting "longstanding" EPA policy concerning the standards by which toxicity information should be reviewed for purposes of §8(e) compliance. Regulatee recognizes that experience with the 1978 Statement of Interpretation may cause a review of its criteri. Regulatee supports and has no objection to the Agency's amending reporting criteria *provided that* such amendment is not applied to the regulated community in an unfair way. However, with the unilateral announcement of the CAP under the auspices of an OCM enforcement proceeding, EPA has wrought a terrific unfairness since much of the criteria EPA has espoused in the June 1991 Reporting Guide and in the Agency's April 2, 1992 amendment is new criteria which does not.exist in the 1978 Statement of Interpretation and Enforcement Policy.

The following examples of new criteria contained in the "Reporting Guide" that is not contained in the Statement of Interpretation follow:

o even though EPA expressly disclaims each "status report" as being preliminary evaluations that should not be regarded as final EPA policy or intent[4], the "Reporting Guide" gives the "status reports" great weight as "sound and adequate basis" from which to determine mandatory reporting obligations. ("Guide" at page 20).

o the "Reporting Guide" contains a matrix that establishes new numerical reporting "cutoff" concentrations for acute lethality information ("Guide" at p. 31). Neither this matrix nor the cutoff values therein are contained in the Statement of Interpretation. The regulated community was not made aware of these cutoff values prior to issuance of the "Reporting Guide" in June, 1991.

othe "Reporting Guide" states new specific definitional criteria with which the Agency, for the first time, defines as 'distinguishable neurotoxicological effects'; such criteria/guidance not expressed in the 1978 Statement of Interpretation.[5];

othe "Reporting Guide" provides new review/ reporting criteria for irritation and sensitization studies; such criteria not previously found in the 1978 Statement of Interpretation/Enforcement Policy.

othe "Reporting Guide" publicizes certain EPA Q/A criteria issued to the Monsanto Co. in 1989 which are not in the Statement of Interpretation; have never been published in the Federal Register or distributed by the EPA to the Regulatee. Such Q/A establishes new reporting criteria not previously found in the 1978 Statement of Interpretation/Enforcement Policy .

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[4]The 'status reports' address the significance, if any, of particular information reported to the Agency, rather than stating EPA's interpretation of §8(e) reporting criteria. In the infrequent instances in which the status reports contain discussion of reportability, the analysis is invariably quite limited, without substantial supporting scientific or legal rationale.

[5] *See, e.g,* 10/2/91 letter from Du Pont to EPA regarding the definition of 'serious and prolonged effects' as this term may relate to transient anesthetic effects observed at lethal levels; 10/1/91 letter from the American Petroleum Institute to EPA regarding clarification of the Reporting Guide criteria.

In discharging its responsibilities, an administrative agency must give the regulated community fair and adequate warning to as what constitutes noncompliance for which penalties may be assessed.

> Among the myriad applications of the due process clause is the fundamental principle that statutes and regulations which purport to govern conduct must give an adequate warning of what they command or forbid.... Even a regulation which governs purely economic or commercial activities, if its violation can engender penalties, must be so framed as to provide a constitutionally adequate warning to those whose activities are governed.

Diebold, Inc. v. Marshall, 585 F.2d 1327, 1335-36 (D.C. Cir. 1978). *See also*, Rollins Environemntal Services (NJ) Inc. v. U.S. Environmental Protection Agency, 937 F. 2d 649 (D.C. Cir. 1991).

While neither the are rules, This principle has been applied to hold that agency 'clarification', such as the Statement of Interpretation, the "Reporting Guide" nor the April 1992 amendments will not applied retroactively.

> ...a federal court will not retroactively apply an unforeseeable interpretation of an administrative regulation to the detriment of a regulated party on the theory that the post hoc interpretation asserted by the Agency is generally consistent with the policies underlying the Agency's regulatory program, when the semantic meaning of the regulations, as previously drafted and construed by the appropriate agency, does not support the interpretation which that agency urges upon the court.

Standard Oil Co. v. Federal Energy Administration, 453 F. Supp. 203, 240 (N.D. Ohio 1978), aff'd sub nom. Standard Oil Co. v. Department of Energy, 596 F.2d 1029 (Em. App. 1978):

The 1978 Statement of Interpretation does not provide adequate notice of, and indeed conflicts with, the Agency's current position at §8(e) requires reporting of all 'positive' toxicological findings without regard to an assessment of their relevance to human health. In accordance with the statute, EPA's 1978 Statement of Interpretation requires the regulated community to use scientific judgment to evaluate the significance of toxicological findings and to determining whether they reasonably support a conclusion of a substantial risk. Part V of the Statement of Interpretation urges persons to consider "the fact or probability" of an effect's occurrence. Similarly, the 1978 Statement of Interpretation stresses that an animal study is reportable only when "it contains reliable evidence ascribing the effect to the chemical." 43 Fed Reg. at 11112. Moreover, EPA's Statement of Interpretation defines the substantiality of risk as a function of both the seriousness of the effect and the probability of its occurrence. 43 Fed Reg 11110 (1978). Earlier Agency interpretation also emphasized the "substantial" nature of a §8(e) determination. See 42 Fed Reg 45362, 45363

(1977). [Section 8(e) findings require "extraordinary exposure to a chemical substance...which critically imperil human health or the environment"].

The recently issued "Reporting Guide" and April 1992 Amendment guidance requires reporting beyond and inconsistent with that required by the Statement of Interpretation. Given the statute and the Statement of Interpretation's explicit focus on substantial human or environmental risk, whether a substance poses a "substantial risk" of injury requires the application of scientific judgment to the available data on a case-by-case basis.

If an overall weight-of-evidence analysis indicates that this classification is unwarranted, reporting should be unnecessary under §8(e) because the available data will not "reasonably support the conclusion" that the chemical presents a substantial risk of serious adverse consequences to human health.

Neither the legislative history of §8(e) nor the plain meaning of the statute support EPA's recent lowering of the reporting threshold that TSCA §8(e) was intended to be a sweeping information gathering mechanism. In introducing the new version of the toxic substances legislation, Representative Eckhart included for the record discussion of the specific changes from the version of H. R. 10318 reported by the Consumer Protection and Finance Subcommittee in December 1975. One of these changes was to modify the standard for reporting under §8(e). The standard in the House version was changed from "causes or contributes to an unreasonable risk" to "causes or significantly contributes to a substantial risk". This particular change was one of several made in TSCA §8 to avoid placing an undue burden on the regulated community. The final changes to focus the scope of Section 8(e) were made in the version reported by the Conference Committee.

The word "substantial" means "considerable in importance, value, degree, amount or extent". Therefore, as generally understood, a "substantial risk" is one which will affect a considerable number of people or portion of the environment, will cause serious injury and is based on reasonably sound scientific analysis or data. Support for the interpretation can be found in a similar provision in the Consumer Product Safety Act. Section 15 of the CPSA defines a "substantial product hazard" to be:

> "a product defect which because of the pattern of defect, the number of defective products distributed in commerce, the severity of the risk, or otherwise, creates a substantial risk of injury to the public."

Similarly, EPA has interpreted the word 'substantial' as a quantitative measurement. Thus, a 'substantial risk' is a risk that can be quantified, *See,* 56 Fed Reg 32292, 32297 (7/15/91). Finally, since information pertinent to the exposure of humans or the environment to chemical substances or mixtures may be obtained by EPA through Sections 8(a) and 8(d) regardless of the degree of potential risk, §8(e) has specialized function. Consequently, information subject to §8(e) reporting should be of a type which would lead a reasonable man to conclude that some type action was required immediately to prevent injury to health or the environment.

Attachment

Comparison:

Reporting triggers found in the 1978 "Statement of Interpretation/ Enforcement Policy",43 Fed Reg 11110 (3/16/78) and the June 1991 *Section 8(e) Guide.*

TEST TYPE	1978 POLICY CRITERIA EXIST?	New 1991 GUIDE CRITERIA EXIST?
ACUTE LETHALITY		
Oral	N}	Y}
Dermal	N}	Y}
Inhalation (Vapors)	}[6]	}[7]
aerosol	N}	Y}
dusts/ particles	N}	Y}
SKIN IRRITATION	N	Y[8]
SKIN SENSITIZATION (ANIMALS)	N	Y[9]
EYE IRRITATION	N	Y[10]
SUBCHRONIC (ORAL/DERMAL/INHALATION)	N	Y[11]
REPRODUCTION STUDY	N	Y[12]
DEVELOPMENTAL TOX	Y[13]	Y[14]

[6] 43 Fed Reg at 11114, comment 14:

"This policy statements directs the reporitng of specifiec effects when unknown to the Administrator. Many routine tests are based on a knowledge of toxicity associated with a chemicalL unknown effects occurring during such a range test may have to be reported if they are those of concern tot he Agency and if the information meets the criteria set forth in Parts V and VII."

[7] Guide at pp.22, 29-31.

[8] Guide at pp-34-36.

[9] Guide at pp-34-36.

[10] Guide at pp-34-36.

[11] Guide at pp-22; 36-37.

[12] Guide at pp-22

[13] 43 Fed Reg at 11112

"Birth Defects" listed.

[14] Guide at pp-22

NEUROTOXICITY	N	Y[15]
CARCINOGENICITY	Y[16]	Y[17]
MUTAGENICITY		
In Vitro	Y}[18]	Y} [19]
In Vivo	Y}	Y}
ENVIRONMENTAL		
Bioaccumulation	Y}	N
Bioconcentration	Y}[20]	N
Oct/water Part. Coeff.	Y}	N
Acute Fish	N	N
Acute Daphnia	N	N
Subchronic Fish	N	N
Subchronic Daphnia	N	N
Chronic Fish	N	N
AVIAN		
Acute	N	N
Reproductive	N	N
Reprodcutive	N	N

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[15] Guide at pp-23; 33-34.

[16] 43 Fed Reg at 11112
"Cancer" listed

[17] Guide at pp-21.

[18] 43 Fed Reg at 11112; 11115 at Comment 15
"Mutagenicity" listed/ *in vivo* vs *invitro* discussed; discussion of "Ames test".

[19] Guide at pp-23.

[20] 43 Fed Reg at 11112; 11115 at Comment 16.

CAS# 83-01-2; 133-55-1

Chem: Diphenylcarbamyl chloride; diphenylcarbamyl azide; N,N'-dimethyl-N,N'-dinitrosoterephthalamide; N,N'-dimethylterephthalamide

Title: Toxicity of diphenylcarbamyl chloride, diphenylcarbamyl azide, N,N'-dimethyl-N,N'-dinitrosoterephthalamide (NTA) and N,N'-dimethylterephthalamide

Date: 7/23/56

Summary of Effects: NTA: fibrous plural adhesions; diphenylcarbamyl chloride: kidney damage in acute study; N,N'-dimethyl-N,N'-dinitrosotcrephalamide: skin sensitizer

TOXICITY OF DIPHENYLCARBAMYL CHLORIDE, DIPHENYLCARBAMYL AZIDE, N,N'-DIMETHYL-N,N'-DINITROSOTEREPHTHALAMIDE (NTA) AND N,N'-DIMETHYLTEREPHTHALAMIDE

Medical Research Projects MR-170.004 and MR-357.001

Report No. 29-56

At the request of the Explosives Department, toxicity tests have been carried out on diphenylcarbamyl chloride (Haskell No. 844), diphenylcarbamyl azide (Haskell No. 845), N,N'-dimethyl-N,N'-dinitrosoterephthalamide (NTA; Haskell Nos. 846 and 1710) and N,N'-dimethylterephthalamide (Haskell No. 1709).

Diphenylcarbamyl azide and NTA are products of the Explosives Department intended for use as blowing agents in the manufacture of sponge rubber and plastic sponge. Diphenylcarbamyl chloride and N,N'-dimethylterephthalamide are the intermediates from which diphenylcarbamyl azide and N,N'-dimethyl-N,N'-dinitrosophthalamide are prepared, respectively.

N,N'-Dimethyl-N,N'-dinitrosoterephthalamide (NTA)

Acute Oral Toxicity

The Approximate Lethal Dose (ALD) for male albino rats was found to be 4275 mg/kg body weight when a 10 to 15 per cent aqueous suspension of the chemical was administered by stomach tube. When the surviving rats were killed eleven days after treatment, acute gastritis and ulceration of the stomach were found in animals which received doses as low as 480 mg/kg.

Subacute Oral Toxicity

Six male albino rats given ten doses of 855 mg/kg/day over a two-week period all survived but showed marked weight loss during treatment; however, when treatment was stopped they all gained satisfactorily. Two of the three animals killed eleven days after the last treatment showed evidence of healed stomach injury. Those killed four days after treatment showed evidence of healing liver injury as well as stomach injury.

Primary Irritancy and Sensitization

Skin irritation and allergic skin sensitization tests were carried out on guinea pigs. NTA was markedly irritating to the intact skin and even a 3.8 per cent solution in acetone produced mild irritation. A 1.7 per cent solution was practically nonirritating to intact skin but strongly irritating when applied to abraded skin. A solution as low in strength as 0.02 per cent was mildly irritating to abraded skin. Reapplication of the material after

seven sensitizing treatments and a two-week rest period indicated that the compound had produced allergic skin sensitization.

Toxicity by Intratracheal Insufflation

In order to study the effect of NTA on lung tissue, experiments were done with guinea pigs in which a suspension of N,N'-dimethyl-N,N'-dinitrosoterephthalamide in physiological saline solution was insufflated into the lungs by injection into the trachea. In one experiment, fourteen adult guinea pigs were used, and the amount of material insufflated was 1 ml of a suspension containing about 13 mg of the test chemical. Twelve of the fourteen guinea pigs were dead within 2[illegible] hours. In each case the cause of death was diagnosed as acute pulmonary edema. The thirteenth guinea pig died 25 days after treatment and was found to have extensive pneumonia and pleural adhesions. The fourteenth guinea pig was sacrificed 30 days after treatment and fibrous pleural adhesions were found in the lungs.

Fourteen other guinea pigs were treated by intratracheal insufflation of a suspension of the test chemical in physiological saline. One milliliter of the suspension was used with each pig and the dose was approximately 1.2 mg of test chemical. All animals survived the treatment and half of the group was sacrificed 30 days after treatment. Six of the seven animals showed pathological changes in the lungs. These consisted of pneumonia and atalectasis. The other seven animals were sacrificed 62 days after treatment. Here again, five of the seven animals showed pathological changes in the lungs consisting of atalectasis, focal pneumonia, and in one case, fibrous pleural adhesions. Although spontaneous pneumonia does occur in guinea pigs, it is believed that most of the changes observed were due to the test chemical.

Sponge Extracts

Acetic acid extracts of polyvinyl chloride sponges blown with NTA were tested on guinea pigs for skin irritation and sensitization. The test results were negative, indicating that there was not enough NTA present to produce a reaction.

Diphenylcarbamyl Azide

Acute Oral Toxicity

The oral ALD of diphenylcarbamyl azide for male albino rats was found to be 7500 mg/kg of body weight when a 20 per cent suspension of the test chemical in peanut oil was administered by stomach tube. Lethal doses produced marked discomfort, weakness, and labored respiration. Gross and micropathological examination of animals given lethal doses of the material revealed acute toxic injury of the liver with swollen vacuolated cells around the portal spaces. Sublethal doses produced slight discomfort, but no pathological changes were found when the rats were sacrificed 9 or 11 days after treatment.

Subacute Oral Toxicity

When doses of 1500 mg/kg of body weight were administered to six rats five times a week for two weeks, the rats survived but lost weight during treatment. However, when treatment was stopped, the rats gained weight. Those animals killed ten days after treatment was stopped compared favorably with control animals, but examination of those killed only four days after treatment revealed a significant enlargement of the liver.

Primary Irritancy and Sensitization Properties

A 25 per cent alcohol-and-acetone solution of diphenylcarbamyl azide was only mildly irritating to both intact and abraded skin of albino guinea pigs. Reapplication of the material after six sensitizing treatments and a two-week rest period indicated that the compound did not cause allergic skin sensitization.

Inhalation Toxicity

Male albino rats survived exposures for six or seven hours to nominal concentrations of 4.0 mg of diphenylcarbamyl azide per liter of air; nor was the compound found to be lethal to two rats exposed to nominal concentrations averaging 0.9 mg/lit of air for four successive days, with a total exposure time of 23 hours. One of the two rats studied in the four-day exposure series showed evidence of bronchopneumonia at autopsy, but this may have been coincidental.

Diphenylcarbamyl Chloride

Acute Oral Toxicity

The oral ALD of diphenylcarbamyl chloride for male albino rats was found to be 1500 mg/kg body weight when the material was administered by stomach tube as a 20 per cent suspension in peanut oil. Lethal doses produced discomfort and death within 1-2 days after treatment. Gross and micropathological examination of the animals revealed that kidney damage was caused by single doses of 1000 mg/kg or more; there was a large amount of albuminous fluid found in the tubules.

Subacute Oral Toxicity

All six of the male albino rats tested survived ten doses of 300 mg/kg/day given over a two-week period. Gross and micropathological examination of the animals revealed enlargement of the kidneys with crystalline deposits in the collecting tubules in two of the three animals sacrificed ten days after the last treatment, and mild tubular nephritis in one of the three animals sacrificed four days after the last treatment.

Primary Irritancy and Sensitization

A 25 per cent acetone solution was nonirritating to the intact skin but was very irritating to abraded skin; concentrations of 0.1 and 1.0 per cent applied to abraded skin produced mild to moderate irritation respectively. Tests for sensitization after six treatments and a two-week rest period, indicated that the guinea pigs had become sensitized to diphenylcarbamyl chloride. Cross-sensitization tests showed that the animals sensitized to diphenylcarbamyl chloride did not respond when challenged with diphenylcarbamyl azide.

N,N'-Dimethylterephthalamide

Primary Irritancy and Sensitization Properties

A 50 per cent suspension of the test chemical in 1 per cent aqueous Duponol PT was found to be mildly irritating to the intact skin of young guinea pigs and very mildly irritating or completely non-irritating to the intact skin of older animals. When tested on abraded skin, the 50 per cent suspension produced a mild to moderate irritation with young guinea pigs but no irritation with older animals. When tested for sensitization after nine sensitizing treatments and a two-week rest period, the reaction indicated that the chemical did not produce an allergic contact dermatitis in any of the treated animals.

Toxicity by Intratracheal Insufflation

Using guinea pigs a suspension of dimethylterephthalamide in physiological saline solution was injected into the trachea. Thirteen guinea pigs were used, and the dose of test chemical was about 33 mg given in 1 ml of aqueous suspension. Six of the animals were sacrificed 30 days after treatment and in only one case was injury to the lung discovered at autopsy. In this case there was minimal atalectasis. The other seven animals were sacrificed 62 days after treatment. Such pathological findings as were made were believed to be coincidental and not attributable to the test chemical.

Summary and Conclusions

N,N'-Dimethyl-N,N'-dinitrosophthalamide (NTA) was found to be of a low order of acute oral toxicity with an ALD of 4275 mg/kg for male albino rats. On the other hand, the compound was shown to be a powerful primary skin irritant and an allergic sensitizer. Due caution should be exercised to avoid skin contact with this substance. When the response of the lung to intratracheal insufflation of NTA was studied, it was found that introduction of about 13 mg per animal caused the death of 12 of 14 guinea pigs within 24 hours due to pulmonary edema. Even 1.2 mg caused anatomical injury to the lungs, although the animals survived. NTA is, therefore, a strong pulmonary irritant.

Diphenylcarbamyl azide was similarly found to be of a low order of acute oral toxicity, with an ALD of 7500 mg/kg for rats. It was only mildly irritating on skin application and was not found to produce sensitization.

Diphenylcarbamyl chloride was found to have an oral ALD value of 1500 mg/kg for male albino rats. Skin tests showed little irritation with intact skin, but strong irritation of abraded skin. Repeated application produced allergic sensitization.

N,N'-Dimethylterephthalamide was found to be of a low order of toxicity when studied by intratracheal insufflation. It was only mildly irritating on skin application and was not found to produce sensitization.

Tests for skin irritation with extracts of sponges blown with NTA were negative, indicating that insufficient free NTA was present to produce irritation or sensitization.

HASKELL LABORATORY FOR TOXICOLOGY
AND INDUSTRIAL MEDICINE

Report by: Jonathan W. Williams
Jonathan W. Williams
Assistant Director

Approved by: John A. Zapp, Jr.
John A. Zapp, Jr.
Director

JWW:ecd
Report No. 29-56
7/23/56

Triage of 8(e) Submissions

Date sent to triage: 5/28/96 NON-CAP (CAP)

Submission number: 12047A TSCA Inventory: (Y) N (D)

Study type (circle appropriate):

Group 1 - Gordon Cash (1 copy total)

ECO AQUATO

Group 2 - Ernie Falke (1 copy total)

(ATOX) (SBTOX) (SEN) w/NEUR

Group 3 -HERD (1 copy each)

STOX	CTOX	EPI	RTOX	GTOX
STOX/ONCO	CTOX/ONCO	IMMUNO	CYTO	NEUR

Other (FATE, EXPO, MET, etc.): _____

Notes:

☒ This is the **original** 8(e) submission; refile after triage evaluation.

☐ This **original** submission has been **split**; rejoin after triage evaluation.

☐ Other:

Photocopies Needed for Triage Evaluation

entire document: (0) 1 2 3

front section and CECATS: (0) 1 2 3

Initials: JW **Date:** 5/23/96

CECATS\TRIAGE TRACKING DBASE ENTRY FORM

CECATS DATA:
Submission # 8EHQ- 1092 -12047 SEQ. A

TYPE: (INT.) SUPP FLWP

SUBMITTER NAME: E. I. Dupont de Nemours and Company

SUB. DATE: 10/15/92 OTS DATE: 10/27/92 CSRAD DATE: 08/25/95

INFORMATION REQUESTED: FLWP DATE:
0501 NO INFO REQUESTED
0502 INFO REQUESTED (TECH)
0503 INFO REQUESTED (VOL ACTIONS)
0504 INFO REQUESTED (REPORTING RATIONALE)

DISPOSITION:
(0639) REFER TO CHEMICAL SCREENING
(0678) CAP NOTICE

VOLUNTARY ACTIONS:
(0401) NO ACTION REPORTED
0402 STUDIES PLANNED/UNDERWAY
0403 NOTIFICATION OF WORKERS/OTHERS
0404 LABEL/MSDS CHANGES
0405 PROCESS/HANDLING CHANGES
0406 APP./USE DISCONTINUED
0407 PRODUCTION DISCONTINUED
0408 CONFIDENTIAL

CHEMICAL NAME:	CAS#
Diphenylcarbamyl azide	83-01-2
N,N'-dimethylterephthalamide	133-55-1
Unknown	Unknown

INFORMATION TYPE:		P F C
0201	ONCO (HUMAN)	01 02 04
0202	ONCO (ANIMAL)	01 02 04
0203	CELL TRANS (IN VITRO)	01 02 04
0204	MUTA (IN VITRO)	01 02 04
0205	MUTA (IN VIVO)	01 02 04
0206	REPRO/TERATO (HUMAN)	01 02 04
0207	REPRO/TERATO (ANIMAL)	01 02 04
0208	NEURO (HUMAN)	01 02 04
0209	NEURO (ANIMAL)	01 02 04
0210	ACUTE TOX. (HUMAN)	01 02 04
0211	CHR. TOX. (HUMAN)	01 02 04
(0212)	ACUTE TOX. (ANIMAL)	01 02 04
(0213)	SUB ACUTE TOX (ANIMAL)	01 02 04
0214	SUB CHRONIC TOX (ANIMAL)	01 02 04
0215	CHRONIC TOX (ANIMAL)	01 02 04

INFORMATION TYPE:		P F C
0216	EPI/CLIN	01 02 04
0217	HUMAN EXPOS (PROD CONTAM)	01 02 04
0218	HUMAN EXPOS (ACCIDENTAL)	01 02 04
0219	HUMAN EXPOS (MONITORING)	01 02 04
0220	ECO/AQUA TOX	01 02 04
0221	ENV. OCCC/REL/FATE	01 02 04
0222	EMER INCI OF ENV CONTAM	01 02 04
0223	RESPONSE REQEST DELAY	01 02 04
0224	PROD/COMP/CHEM ID	01 02 04
0225	REPORTING RATIONALE	01 02 04
0226	CONFIDENTIAL	01 02 04
0227	ALLERG (HUMAN)	01 02 04
(0228)	ALLERG (ANIMAL)	01 02 04
0239	METAB/PHARMACO (ANIMAL)	01 02 04
0240	METAB/PHARMACO (HUMAN)	01 02 04

INFORMATION TYPE:		P F C
0241	IMMUNO (ANIMAL)	01 02 04
0242	IMMUNO (HUMAN)	01 02 04
0243	CHEM/PHYS PROP	01 02 04
0244	CLASTO (IN VITRO)	01 02 04
0245	CLASTO (ANIMAL)	01 02 04
0246	CLASTO (HUMAN)	01 02 04
0247	DNA DAM/REPAIR	01 02 04
(0248)	PROD/USE/PROC	01 02 04
0251	MSDS	01 02 04
0299	OTHER	01 02 04

TRIAGE DATA: NON-CBI INVENTORY
(YES)
CAS SR
NO
(IN TSCA INV)

ONGOING REVIEW
YES (DROP/REFER)
NO (CONTINUE)
REFER

SPECIES
RAT
GP

TOXICOLOGICAL CONCERN:
LOW
MED
HIGH
} see attached Sheet

USE: Blowing agents in manuf. of sponge rubber and plastic sponge

PRODUCTION:

COMMENTS

=> D RN SY

L33 ANSWER 1 OF 1 REGISTRY COPYRIGHT 1995 ACS
RN ***133-55-1*** REGISTRY
CN 1,4-Benzenedicarboxamide, N,N'-dimethyl-N,N'-dinitroso- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Terephthalamide, N,N'-dimethyl-N,N'-dinitroso- (6CI, 7CI, 8CI)
OTHER NAMES:
CN BL 353
CN N,N'-Dimethyl-N,N'-dinitrosoterephthalamide
CN N,N'-Dinitroso-N,N'-dimethylterephthalamide
CN N,N'-Dinitroso-N,N'-dimethylterephthalic acid amide
CN Nitrosan
CN Nitrosan (blowing agent)

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	116.77	117.03

STN INTERNATIONAL LOGOFF AT 13:48:54 ON 27 APR 95

#12047

N,N'-Dimethyl-N,N'-dinitrosoterephthalamide
M

Dermal sensitization is medium based on allergic skin reactions in guinea pigs (severity and incidence not reported).

M,L

Dermal irritation is medium based on marked irritation in guinea pigs; dermal irritation is low based on mild irritation in guinea pigs exposed to various dilutions (3.8% and 1.7%).

L

Acute oral toxicity is low based on an approximate lethal dose of 4275 mg/kg in rats. Acute gastritis and stomach ulceration were observed at doses of ≥480 mg/kg.

L

Subacute oral toxicity is low based on no deaths in rats exposed to 855 mg/kg for 2 weeks. Healed stomach and liver injury were observed at 11 and 4 days post-exposure, respectively.

L

Intratracheal toxicity is low based on 13/14 deaths in guinea pigs exposed to 13000 mg/kg (assumed density of 1 for conversion of mg/ml to mg/kg) insufflated into the lungs, and 0/14 deaths at 1200 mg/kg. Observations at necropsy included acute pulmonary edema, extensive pneumonia and pleural adhesions at 13000 mg/kg, and pneumonia, atelectasis and pleural adhesions at 1200 mg/kg.

Diphenylcarbamyl Azide
L

Acute oral toxicity is low based on an approximate lethal dose of 7500 mg/kg in rats. Clinical signs including weakness and labored respiration, and pathological changes in the liver were observed in the decedents.

L

Subacute oral toxicity is low based on no deaths in rats exposed to 1500 mg/kg 5 days/week for 2 weeks. Liver enlargement was observed in rats sacrificed 4 days post-exposure.

L

Dermal irritation is low based on mild irritation in guinea pigs.

L

Dermal sensitization is low based on no allergic reactions in guinea pigs.

L

Acute inhalation toxicity is low based on no deaths in rats exposed to 4.0 mg/L for 6 or 7 hours.

L

Subacute inhalation toxicity is low based on no mortality (0/2) in rats exposed to 0.9 mg/L for 4 days; 1/2 rats demonstrated bronchopneumonia at autopsy.

Diphenylcarbamyl Chloride
M

Dermal sensitization is medium based on positive reactions in guinea pigs (no severity or incidence was reported).

L

Dermal irritation is low based on no irritation in guinea pigs.

L
Acute oral toxicity is low based on an approximate lethal dose of 1500 mg/kg in rats. Kidney damage was observed at ≥1000 mg/kg.

L

Subacute oral toxicity is low based on no deaths (0/6) in rats exposed to 300 mg/kg/day for 2 weeks. Pathological changes were observed in the kidneys.

N,N'-Dimethylterephthalamide
Dermal Irritation - Low
Dermal Sensitization - Low

L

Dermal irritation is low based on mild irritation in young and old guinea pigs.

L

Dermal sensitization is low based on no allergic contact dermatitis in guinea pigs. Intratracheal toxicity is low based on no deaths (0/13) in guinea pigs exposed to 33000 mg/kg (density or 1 assumed in conversion of mg/L to mg/kg) by intratracheal insufflation. Minimal atelectasis was observed in 1 animal.